medicated immediately with clarithromycin, 500 mg twice a day, inhaled steroids and bronchodilators. Within 48 hours, all symptoms resolved except the cough, which again became dry. Chest x-ray films showed a left upper lobe infiltrate. A PPD skin test was negative. She remained afebrile until the tenth day of medication, when she had chills, her cough worsened, and a fever of 38.3°C (101°F) developed. The next day, intravenous ceftriaxone sodium was administered. She was admitted to the hospital on day 12 of clarithromycin therapy with a fever to 40°C (104°F). She had recently traveled to an Arizona construction site, and she had many friends who had the acquired immunodeficiency syndrome (AIDS).

On admission the patient was coughing, had chills, and was requesting antipyretics. Her blood pressure was 100/60 mm of mercury, pulse rate 88 beats per minute, respirations 20 per minute, and temperature 40°C. Her leukocyte count was 7.4×10^9 per liter (7,400 per mm^3). A chest x-ray film showed an increase in left upper lobe infiltrates. The clarithromycin was stopped, and a regimen of intravenous erythromycin and cefuroxime was begun. A bronchoscopy showed inflammation and purulence in the left apical posterior bronchus. All stains and cultures of blood, sputum, and bronchoscopy specimens were negative for bacteria, virus, acid-fast bacilli, fungi, and *Chlamydia* and *Legionella* species, as were serologic tests of the same. Isoniazid, rifampin, pyrazinamide, and itraconazole were added on the second day of her hospital stay; however, her pulmonary function deteriorated, her fever escalated, her infiltrates progressed, and oxygen saturation fell into the 80s, so that by hospital day 7, pulmonary infiltrates were described as severe, multisegmental, and bilateral with pleural effusions. The patient had severe dyspnea at rest while receiving 100% fractional inspired oxygen; a Po_2 was 54 mm of mercury with the patient breathing room air.

An open-lung biopsy of the right lower lobe was done on the eighth hospital day. Microscopic studies revealed extensive bronchiolitis with pneumonia compatible with *M pneumoniae* pneumonia. Cold agglutinins returned positive at 1:128. Mycoplasma titers rose from 1:16 to 1:128. A course of parenteral steroids was begun on day 9. Her fever resolved immediately, and her shortness of breath resolved over the next 48 hours. Dramatic improvement was seen on her chest x-ray films within four days. A week later she was home without residual compromise of pulmonary function.

Discussion

Although conventional medical teaching conveys to us that illness caused by the microorganism *Mycoplasma pneumoniae* is mild, the literature is rife with reports of near-fatal and fatal cases of *M pneumoniae* pneumonia.[2-5] The diagnosis for as many as 20% of patients admitted to a hospital for community-acquired pneumonia may be *M pneumoniae* pneumonia.[6] The severity of illness has been attributed to a lack of timely administration of appropriate antibiotics.

The extrapulmonary complications of *M pneumoniae* pneumonia have been attributed to infection in those systems as well as to mechanisms of immune origin.[7] This case lends further support to an immune mechanism as the cause of "pulmonary complications" of *M pneumoniae* pneumonia and for the use of parenteral corticosteroids in the management of severe *M pneumoniae* pneumonia.

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Drs Chan and Welsh Respond

TO THE EDITOR: We thank Lawrence A. Cone, MD, DSc, and colleagues and Linda Dubins, MD, for reporting their cases of severe *Mycoplasma pneumoniae* pneumonia. Both patients, previously healthy and relatively young, improved only after the administration of corticosteroids. In addition, in the case reported by Dubins, progressive *M pneumoniae* pneumonia developed despite immediate treatment with clarithromycin. These findings are consistent with those of our patient and of the cases we reviewed.[1] Based on our arbitrary but strict criteria for respiratory failure in our review, the case presented by Dubins would not have been included in our series, despite the severe nature.

Cone and colleagues reported the case of a patient with respiratory failure and disseminated intravascular coagulation due to *M pneumoniae*. We agree that proinflammatory cytokines are probably involved in the exuberant, and possibly deleterious, inflammatory response. The mechanism of T-cell proliferation with proinflammatory cytokine release in response to a superantigen of *M pneumoniae* proposed by Cone and associates is certainly a possibility. We wonder, however, whether such a response, presumably due to the inflammatory Th1 cytokines interleukin (IL)-2 and gamma interferon, is in fact caused by a relative lack of anti-inflammatory cytokines IL-4 and IL-10 produced by Th2 cells. This hypothesis is indirectly supported by a report that showed that a low IL-10 response in sepsis is associated with an increased incidence of shock.[2] In trying to resolve this apparent paradox of the salutary effect of

anti-inflammation in host defense against microorganisms, our belief is that both an inflammatory response and then an anti-inflammatory "counterregulatory response" are required, but in a sequential fashion. Moreover, the Th2 cytokines are important mediators of the humoral arm of the specific immunity that we believe to be an important host defense mechanism against *M pneumoniae*.[1]

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Hereditary Hemochromatosis—Importance of Age and Sex?

To the Editor: The article by Yutaka Niihara, MD, and colleagues made interesting and informative reading.[1] The authors describe a unique case of hereditary hemochromatosis, which is the most frequently inherited disease in whites, seen in a 33-year-old woman. Typically, hemochromatosis becomes clinically manifest later in life and is seen more frequently in men than in women. In this context, we would like to draw your attention to another interesting case of a young woman found to have an early onset of symptoms associated with hemochromatosis.

Silva and co-workers report the case of a 26-year-old woman who for eight years had amenorrhea, cardiac failure, diabetes mellitus, and increased pigmentation of the skin, with biochemical markers of iron overload.[2] They emphasize that hemochromatosis must be excluded in all patients with unexplained cardiac failure. With early diagnosis and treatment, the life expectancy of such patients can be substantially prolonged.

Whereas perinatal or neonatal hemochromatosis is recognized as a distinct clinical disorder, Kaikov and associates report the cases of three asymptomatic siblings with hereditary hemochromatosis who had elevated serum iron levels, confirmed by hepatic biopsy studies.[3] Repeated phlebotomies resulted in a considerable decline of hepatic iron content. Hence, the diagnosis of hereditary hemochromatosis must be considered more frequently in children; regular phlebotomies may minimize organ dysfunction from iron overload in these patients.

Adams reviewed the cases of 57 families with hereditary hemochromatosis and found pronounced differences in iron overload among HLA-identical, sex-matched siblings.[4] Hence, the rate of iron accumulation may vary, and the extent of iron loading in hereditary hemochromatosis is not solely dependent on the duration of the disease.

These reports, in conjunction with Niihara and colleagues' case, reiterate that, regardless of a patient's age or sex, the diagnosis of hereditary hemochromatosis should be considered in patients with iron overload.

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Drs Niihara, Brouwer, and Cantos Respond

To the Editor: We would like to thank Abhay Anand, MD, and colleagues for their commentary and reminder of a recent report of a young woman with severe hemochromatosis.[1] That report, like ours,[2] stresses the importance of considering the diagnosis of hereditary hemochromatosis, regardless of age or sex, if certain clinical findings are present. Because iron deficiency is prevalent and its prevention is emphasized in our society, iron overload may easily be overlooked. It is our hope that these reports will increase awareness of the disease and improve the recognition and management of patients with hereditary hemochromatosis as well as their family members.

Finally, we would like to emphasize the importance of screening the family members of patients with hereditary hemochromatosis. Although this was not the focus of our recent report,[2] the importance of screening in this particular population cannot be overemphasized.

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